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Relationship between body mass index and arterial stiffness in a health assessment Chinese population

Biwen Tang, MM[a], Fangxiu Luo, MM[b], Jiehui Zhao, MM[c], Jing Ma, MM[d], Isabella Tan, PhD[e], Mark Butlin, PhD[e], Alberto Avolio, PhD[e], Junli Zuo, MD, PhD[a,e,*]

Abstract

Pulse wave velocity (PWV) is a reliable measurement of arterial stiffness. Our study assesses the association between body mass index (BMI) and brachial-ankle PWV (baPWV) in a healthy cohort and seeks to explain possible mechanisms associated with the obesity paradox.

A cross-sectional study was conducted in 578 normal individuals. The mean age was 48.3 ± 14.6 years, and 468 (81.0%) were men. 288 subjects (49.8%) were overweight and obese. baPWV and ankle-brachial index (ABI) were performed to evaluate arterial stiffness and atherosclerosis respectively. Normal weight was defined as 18.5 < BMI <25 kg/m^2, overweight as 25 ≤ BMI < 28 kg/m^2 and obesity as BMI ≥28 kg/m^2.

The overweight/obese subjects had significantly higher baPWV than the normal-weight group (1490.0 ± 308.0/1445.2 ± 245.2 cm/s vs 1371.2 ± 306.4 cm/s, $P < .001$). For the whole cohort, baPWV showed a significant positive correlation with BMI (r = 0.205, $P < .001$). However, baPWV was significantly lower as BMI increased: 1490.0 ± 308.0 cm/s (overweight); 1445.2 ± 245.2 cm/s (obese); $P < .001$) when adjusted for age, gender, heart rate, mean blood pressure, and cardiovascular risk factors (glucose, cholesterol, triglyceride, and low-density lipoprotein). For the whole cohort BMI was negatively associated with baPWV (β = −0.06, $P = .042$). ABI showed no relationship with BMI. In a middle-age healthy Chinese population, arterial stiffness measured as baPWV increased with BMI.

Evidence of reduced arterial stiffness with increasing BMI when accounting for all other cardiovascular risk factors may contribute to underlying factors involved in the obesity paradox that becomes more prominent with increasing age.

Abbreviations: ABI = ankle-brachial index, baPWV = brachial-ankle PWV, BMI = body mass index, BP = blood pressure, CVD = cardiovascular diseases, HR = heart rate, MAP = mean arterial pressure, PWV = Pulse wave velocity, SAD = sagittal abdominal diameter, WC = waist circumference, WHtR = waist/height ratio.

Keywords: arterial stiffness, body mass index, brachial–ankle pulse wave velocity (baPWV), obesity paradox

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[a] Department of Geriatric Medicine, [b] Department of Pathology, Shanghai Jiaotong University School of Medicine, [c] Daning Community Health Service Center, [d] Department of Health Assessment, Ruijin Hospital North, Shanghai Jiao Tong School of Medicine, Shanghai, China, [e] Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Macquarie University, Sydney, Australia.

[*] Correspondence: Junli Zuo, Department of Geriatric Medicine, Ruijin Hospital North, Shanghai Jiaotong University School of Medicine, 9B, No. 999, Xiwang Road, Shanghai, China (e-mail: zuo-junli@163.com).

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1. Introduction

Obesity has become a worldwide health problem. Obesity has risen dramatically in developing countries; it can lead to a range of physical and psychosocial health problems. The Global Burden of disease Study 2013 showed that the proportion of adults being overweight or obese increased between 1980 and 2013 from about 28.8% to 36.9% in men and 29.8% to 38% in women.[1] The incidence of obesity in China ranks second in the world.[2] The increase in obesity has an important impact on health and reduced quality of life.[3,4] Obesity has led to an increase in prevalence of cardiovascular diseases (CVD) such as hypertension, coronary heart disease, heart failure, atrial fibrillation, type 2 diabetes, dyslipidemia, and sleep apnea.[2,5–8] However, many recent studies have shown surprisingly good prognosis among overweight and at least mildly obese patients with CVD, the so-called obesity paradox.[5,7] This is notwithstanding obesity being an independent risk factor for many cardiovascular diseases. For patients with cardiovascular disease, the long-term prognosis of obese populations is often better than lean populations.[5,9] This is the obesity paradox. Pulse wave velocity (PWV) has been shown to be a reliable measurement of arterial stiffness; it has been shown to be an independent maker of CVD and mortality.[10,11] Our study assesses the association between BMI and baPWV in a healthy cohort and seeks to clarify possible mechanisms associated with the obesity paradox.

2. Patients and methods

This is a cross-sectional study conducted in 578 normal individuals attending a health assessment clinic without taking any antihypertensive medication at Ruijin Hospital North, Shanghai, from January 2017 to June 2018. Subjects were divided into 3 groups on the basis of their body mass index (BMI) ($18.5 < BMI < 24\,kg/m^2$ normal, 24–28 kg/m^2 overweight, ≥ 28 kg/m^2 obese) according to Chinese Guidelines for the Prevention and Control of Overweight and Obesity in Adults and 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults.[12,13] Body height and weight were measured without shoes using a portable stadio meter. BMI was calculated as the weight in kilograms divided by height in meters squared (kg/m^2). The basic information of the patients was extracted from the participants' medical records. All subjects provided written informed consent. The Ethics Committee of Ruijin Hospital North, Shanghai, approved the study protocol.

Noninvasive measurements of peripheral blood pressure (BP) and brachial-ankle PWV (baPWV) were performed in the supine position using the Omron device (BP-203RPEIII VP-1000 Kyoto, Japan). After the subject had rested at least 10 minutes in the supine position, the pressure cuffs were wrapped on both upper arms and ankles. The device simultaneously and automatically measured blood pressure in both ankles and upper arms. Ankle-brachial index (ABI) was determined by the device as the ratio of the systolic blood pressures at the ankle to the systolic blood pressures in the upper arm (brachial).

2.1. Statistical analysis

Continuous variables are presented as mean ± SD. Pearson test was used to evaluate the correlation between normally distributed univariate variables and BMI. A two-sided P value less than .05 was considered statistically significant throughout the analyses. The association of BMI with baPWV was assessed by means of linear regression. The analyses were performed using SPSS, version 17.0 (SPSS, Chicago, IL).

3. Results

Table 1 shows clinical characteristics of the study population by BMI. The mean age was 48.3 ± 14.6 years, and 468 (81.0%) were men. Three hundred sixty nine subjects (50.2%) had normal weight, 146 subjects (25.3%) were overweight and 63 subjects (10.9%) were obese. Fasting glucose, uric acid level of lipid metabolism and blood pressure were significantly higher in overweight/obesity subjects than in the normal-weight group. The overweight/obesity subjects had significantly higher baPWV than the normal-weight group (1490.0 ± 308.0/1445.2 ± 245.2 cm/s vs 1371.2 ± 306.4 cm/s, $P < .001$). There were no significant differences in heart rate (HR) and ABI between the normal-weight and overweight/obesity group ($P > .05$).

Pearson correlation showed BMI was significantly and positively correlated with baPWV ($r = 0.205$, $P < .001$), systolic blood pressure ($r = 0.311$, $P < .001$), diastolic blood pressure ($r = 0.344$, $P < .001$) and mean arterial pressure (MAP) ($r = 0.371$, $P < .001$), but there was no significant correlation between BMI and ABI ($r = 0.116$, $P > .05$). (Table 2).

However, baPWV was significantly lower as BMI increased (baPWV 1418 ± 187, 1409 ± 180, 1345 ± 182 cm/s; $P = .023$) in the 3 groups adjusted for MAP and cardiovascular risk factors (Fig. 1).

Table 3 shows the predictors of baPWV, with baPWV considered as the dependent variable. When BMI and age were included as continuous variables, age, male gender, HR, and MAP were all significantly associated with baPWV. BMI did not have association with baPWV but showed a negative beta coefficient ($\beta = -0.04$, $P = .149$). Further models also did not find any association between BMI and baPWV. With BMI and age as

Table 1

Characteristics of the study population.

	Total	Normal-weight (18.5–25)	Overweight (25–28)	Obesity (BMI ≥28 kg/m²)	*P* value
	578	369	146	63	
Age (years)	48.3 ± 14.6	46.7 ± 14.5	51.9 ± 14.4**	49.8 ± 14.7	<.001
Sex (male: female)	468: 110	273: 96	136: 10	59: 4	<.001
BMI (kg/m²)	24.2 ± 3.0	22.4 ± 1.7	26.3 ± 0.9**	29.7 ± 1.7**	<.001
eGFR (ml/min/1.73 m²)	89.3 ± 13.0	89.9 ± 12.6	88.0 ± 14.2	89.4 ± 11.8	.381
UA (umol/L)	372.1 ± 83.4	351.9 ± 77.9	396.0 ± 75.5**	435.0 ± 87.6**	<.001
FPG (mmol/L)	5.4 ± 1.2	5.1 ± 1.0	5.8 ± 1.4**	5.8 ± 1.5**	<.001
TC (mmol/L)	3.8 ± 1.9	3.7 ± 1.8	3.8 ± 1.9	4.4 ± 2.3*	.043
TG (mmol/L)	3.1 ± 2.6	2.8 ± 2.0	3.2 ± 1.9*	4.3 ± 5.4**	<.001
HDL (mmol/L)	1.2 ± 0.3	1.3 ± 0.3	1.2 ± 0.2**	1.1 ± 0.2**	<.001
LDL (mmol/L)	3.2 ± 0.7	3.1 ± 0.7	3.2 ± 0.7	3.4 ± 0.9**	.023
HbA1c (%)	5.8 ± 0.8	5.7 ± 0.6	6.0 ± 0.8**	6.1 ± 1.2**	<.001
SBP (mm Hg)	133.9 ± 23.0	129.4 ± 21.6	140.6 ± 22.1**	145.0 ± 25.6**	<.001
DBP (mm Hg)	77.8 ± 11.9	75.5 ± 11.0	81.0 ± 12.1**	84.2 ± 11.7**	<.001
MAP (mm Hg)	96.9 ± 14.2	93.8 ± 13.3	101.2 ± 13.7**	104.8 ± 15.2**	<.001
HR (b.p.m)	71.2 ± 10.9	71.2 ± 10.8	70.4 ± 11.1	73.1 ± 10.9	.264
baPWV (cm/s)	1409.3 ± 304.8	1371.2 ± 306.4	1490.0 ± 308.0**	1445.2 ± 245.2	<.001
ABI	1.14 ± 0.10	1.13 ± 0.09	1.16 ± 0.11	1.15 ± 0.12	.048

Values are expressed as mean ± SD.

* $P < .05$with normal group.

** $P < .001$with normal group.

ABI = ankle brachial index, baPWV = brachial ankle pulse wave velocity, BMI = body mass index, DBP = diastolic blood pressure, eGFR = estimated glomerular filtration rate, FPG = Fasting plasma glucose, HDL = high density lipoprotein cholesterol, HR = heart rate, LDL = low density lipoprotein cholesterol, MAP = mean arterial pressure, SBP = systolic blood pressure, TC = cholesterol TG = triglycerides, UA = uric acid.

Table 2

Pearson correlation among variables.

	BMI	SBP	DBP	MAP	baPWV	ABI
BMI						
SBP	0.311**					
DBP	0.344**	0.601**				
MAP	0.371**	0.883**	0.883**			
baPWV	0.205**	0.636**	0.383**	0.567**		
ABI	0.116	0.350**	0.184**	0.283**	0.297**	

ABI = ankle brachial index, baPWV = brachial ankle pulse wave velocity, BMI = body mass index, DBP = diastolic blood pressure, MAP = mean arterial pressure, SBP = systolic blood pressure.
** $P < .001$.

categorical variables, the study showed that age >50 years was positively associated with baPWV, whereas BMI ≥25 kg/m^2, was not associated with baPWV (β = -0.021, $P = .477$) when adjusted for age (≥50 vs <50 years), sex, BMI (≥25 vs <25 kg/m^2), HR, and MAP. When adjusting for glucose, cholesterol, triglyceride, and low-density lipoprotein, BMI showed a negative association with baPWV (β = −0.06, $P = .042$).

4. Discussion

In our study of a healthy cohort undergoing routine health check, we found that BMI has a positive association with baPWV but is not an independent predictor of arterial stiffness. However, when accounting for age, blood pressure, and cardiovascular risk factors, a significant negative relation was found between baPWV and BMI, suggesting that, in this population, there may be evidence of the potential benefit of obesity in relation to arterial stiffness, as a possible mechanism associated with the obesity paradox, although it is blunted by the presence of other factors of cardiovascular risk.

The mean age of our study population was 48.3 ± 14.6 years (81.0% men). The overweight/obese subjects had significantly higher baPWV than the normal-weight group. They also had higher glucose, uric acid, level of lipid metabolism, and blood pressure. BMI showed a significant positive correlation between baPWV and blood pressure.

There are many speculations for the obesity paradox.[14–16] For example, obese people have higher cardiovascular and lung metabolic reserves, adipose tissue may produce specific hormones and cytokines, and they also have reduced sympathetic activation. Although obesity may be an important factor for arterial remodeling, resulting in hemodynamic and arterial changes that are detrimental to vascular function,[17] vascular endothelial function in obese individuals may still be normal.[18]

PWV has been extensively used as a noninvasive measure of arterial stiffness. PWV can predict adverse cardiovascular outcomes and all-cause mortality in unselected populations.[19,20] The correlation between obesity and PWV is still controversial, and whether it is 1 of the determining mechanisms of obesity paradox remains to be studied.

A previous Brazilian study[21] suggested that BMI was negatively correlated with PWV and this correlation was independent of blood pressure. This finding suggested that that obesity was a protective factor, and obese patients had lower arterial stiffness. It may be 1 of the mechanisms of the obesity paradox. However, some other studies[22–24] suggested that BMI

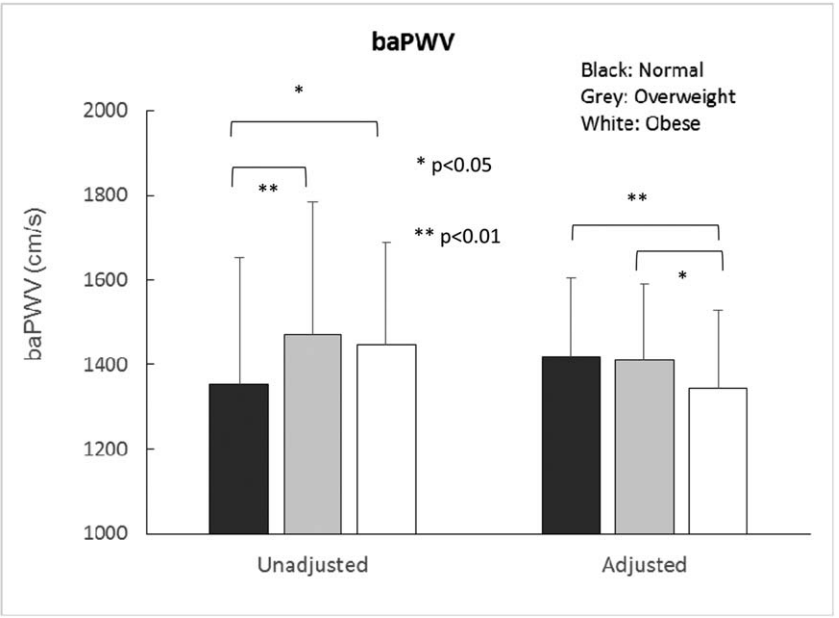

Figure 1. Comparison of change in baPWV with BMI in the 3 groups (normal weight, overweight, obese) when unadjusted and adjusted for cardiovascular risk factors.

Table 3

Determinants of baPWV.

Variable	B	β	*P* value	R^2
Moldel 1				0.643
(Constant)	−22.48			
Age	12.242	0.587	.000	
Sex (Female vs male)	−55.41	−0.071	.009	
HR	5.041	0.180	.000	
BMI	−4.05	−0.040	.149	
MAP	6.65	0.311	.000	
Moldel 2				0.645
(Constant)	−90.44			
Age	12.26	0.588	.000	
Sex (Female vs male)	−50.98	−0.066	.014	
HR	5. 03	0.179	.000	
BMI (≥25 kg/m^2 vs <25kg/m2)	−16.92	−0.027	.312	
MAP	6.53	0.305	.000	
Moldel 3				0.536
(Constant)	291.44			
Age (≥50 vs <50)	279.79	0.456	.000	
Sex (Female vs male)	−79.17	−0.102	.001	
HR	5.12	0.808	.000	
BMI (≥25 kg/m^2 vs <25 kg/m^2)	−13.59	−0.021	.477	
MAP	7.67	0.358	.000	
Moldel 4				0.552
(constant)	202.36			
Age (≥50 vs <50)	248.74	0.387	.001	
Sex (Female vs male)	−78.08	−0.096	.001	
HR	5.084	0.173	.000	
BMI (≥25 kg/m^2 vs <25 kg/m^2)	−30.682	−0.046	.132	
MAP	6.982	0.310	.000	
Glu	48.971	0.181	.000	
TC	−12.504	−0.075	.026	
TG	2.679	0.022	.519	
LDL	−0.383	−0.001	.980	
Model 5				0.651
(constant)	−53.27			
Age	12.57	0.58	.000	
Sex (Female vs male)	−56.28	−0.07	.011	
HR	5.14	0.18	.000	
BMI	−6.15	−0.06	.042	
MAP	6.79	0.30	.000	
Glu	27.48	0.10	.000	
TC	3.43	0.02	.503	
TG	−2.23	−0.02	.547	
LDL	−24.59	−0.06	.069	

BMI = body mass index, Glu = glucose, HR = heart rate, LDL = low density lipoprotein, MAP = mean arterial pressure, TC = cholesterol, TG = triglyceride.

was positively correlated with PWV, suggesting that obesity accelerates the progression of arterial stiffness. Another study[11] showed that obesity has no correlation with arterial stiffness. The above differences may be related to the following factors:

1. *Sample size*. Our study included a small sample size and may have a certain impact on the results.
2. *Blood pressure*. PWV is related to the pressure-dependent effects of stiffness of the arterial wall; hence PWV is associated with blood pressure. There are suggestions that studies showing a positive correlation between BMI and PWV did not consider the effect of blood pressure on the correlation between BMI and PWV.[25] Our study suggested that MAP had a significant impact on PWV, but the interaction of relevant risk factors remains to be further studied.
3. *Metabolic status effects*. Obesity may also have certain benefits while bringing many adverse effects. For example, obese people have higher nutritional reserves and can cope with acute stress events and increased metabolic needs.[26] Adipose tissue produces beneficial hormones and cytokines.[27] Studies have confirmed that obese insulin-sensitive individuals have a favorable metabolic profile compared to the obese insulin-resistant subjects.[28] The interaction of these risks and benefits may create uncertainty regarding the correlation between BMI and PWV.
4. *Abdominal obesity*. Previous studies have suggested that abdominal obesity (waist/height ratio, (WHtR) and waist circumference),[29] sagittal abdominal diameter (SAD)[28] is more strongly associated with PWV than BMI. Another study in 305 individuals showed that with every 0.1 point increase in WHtR and every cm increase in waist circumference (WC), the PWV increased 0.041 and 0.029 m/second, WHtR and WC are better than BMI when considering arterial stiffness.[29] The above results suggest that the type of obesity may have a greater impact on PWV.

When arterial stiffness was assessed using baPWV, our study showed that age, HR, and MAP were positively correlated with baPWV, and gender was negatively correlated with PWV. The effect of blood pressure on PWV has been discussed above.

Our study also suggests that gender has a significant impact on PWV. Compared with men, women have particular hemodynamic characteristics, such as estrogen-mediated vascular relaxation, shorter length of the arterial tree, difference of fat distribution, and HR.[25] The presence of adipose tissue and its related hormones may have a special impact on female hemodynamics. Group studies can be conducted to identify gender differences in the future. The significant positive correlation between HR and baPWV, which is consistent with the Brazilian study[21] on obesity, suggests an increase in sympathetic tone, or an increase in aortic wall stress, thereby increasing PWV.[30]

There are several limitations to be acknowledged in the current study. First, the study was performed by cross-sectional design, and so cannot provide the predictive values of BMI on the progression of arterial stiffness. Second, there is lack of data on antihypertensive medication, smoking states, caffeine intake, and medications. Third, we did not assess participants' body composition, such as fat mass, lean mass, skeletal muscle mass, and waist and hip circumference.

In conclusion, age, HR, MAP are positively correlated with PWV, female sex is negatively correlated with PWV. The findings of this study show that arterial stiffness as measured by baPWV is positively associated with increase in BMI but the relationship is negative when corrected for cardiovascular risk factors. This suggests a potential vascular mechanism for the obesity paradox, although the age-related effects of obesity on PWV remains to be further studied in larger and elderly cohorts.

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Author contributions

Conceptualization: Alberto Avolio, Junli Zuo.
Data curation: Biwen Tang, Fangxiu Luo, Jiehui Zhao, Jing Ma.
Formal analysis: Biwen Tang, Fangxiu Luo, Junli Zuo.
Investigation: Jing Ma.
Methodology: Jiehui Zhao, Isabella Tan, Mark Butlin.
Resources: Jing Ma.
Software: Isabella Tan.
Supervision: Mark Butlin, Alberto Avolio, Junli Zuo.
Validation: Mark Butlin.
Writing – original draft: Biwen Tang, Fangxiu Luo, Jiehui Zhao.
Writing – review & editing: Isabella Tan, Alberto Avolio, Junli Zuo.
Junli Zuo orcid: 0000-0002-9850-405X.

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